Nutritional Support for Patients with AKI

Etienne Macedo, MD, PhD, FASN

Outline

- Metabolic and nutritional changes during AKI
- How to evaluate nutritional status in patient with AKI
- What is adequate nutritional support during different phases of AKI
Metabolic Changes During AKI

AKI is associated with systemic stress-induced hormonal and metabolic changes

- Increase resistance to anabolic signals and insulin
- High glucose concentration
- Increased levels of inflammatory mediators

Fiaccadori and Cremaschi, Current Opinion in Critical Care 2009

Metabolic Changes During AKI

Pre-morbid conditions
- DM, cardiovascular disease, infection, aging
- Production of inflammatory cytokines

Severity of the disease state
- Anemia, acidosis, uremia
- Oxidative and carbonyl stress
- Volume overload
- Nutrient intake, presordial dietary restrictions

Process of care
- Nutrient loss during dialysis
- Dialysis treatment related factors, AV graft, dialysis membrane

Fouque et al., KI 2008
Preiser et al., Critical Care 2015
Cuesta JM et al., Crit Care Med 2012
Malnutrition in Kidney Disease

- Increased inflammation
- Increased energy expenditure (EE)
- Stress hyperglycemia
- Negative nitrogen balance

**Protein Energy Wasting Syndrome**

**Definition**

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Biochemical</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Serum chemistry</strong></td>
<td>At least one</td>
</tr>
<tr>
<td>Serum albumin &lt; 3.8 g per 100 mL (Normal range: 3.8 – 5.0 g/dL)</td>
<td></td>
</tr>
<tr>
<td>Serum prealbumin (transferrin) &lt; 30 mg per 100 mL (normal range: 30 – 60 mg/dL)</td>
<td></td>
</tr>
<tr>
<td>Body mass</td>
<td>BMI &lt; 23*</td>
</tr>
<tr>
<td>Unintentional weight loss over time: 5% over 3 months or 10% over 6 months</td>
<td></td>
</tr>
<tr>
<td>Total body fat percentage &lt; 10%</td>
<td></td>
</tr>
<tr>
<td><strong>Muscle mass</strong></td>
<td></td>
</tr>
<tr>
<td>Muscle wasting: reduced muscle mass 5% over 3 months or 10% over 6 months</td>
<td></td>
</tr>
<tr>
<td>Reduced mid-arm muscle circumference area (reduction &gt; 10% in relation to 50th percentile of reference population)</td>
<td></td>
</tr>
<tr>
<td>Creatinine appearance*</td>
<td></td>
</tr>
<tr>
<td><strong>Dietary intake</strong></td>
<td></td>
</tr>
<tr>
<td>Unintentional low DRI &lt; 0.80 g.kg⁻¹.day⁻¹ for at least 2 months* for dialysis patients or &lt; 0.6 g.kg⁻¹.day⁻¹ for patients with CKD stages 2-5</td>
<td></td>
</tr>
<tr>
<td>Unintentional low DEI &lt; 25 kcal.kg⁻¹.day⁻¹ for at least 2 months*</td>
<td></td>
</tr>
</tbody>
</table>

DO NOT COPY
Factors associated with PEW in AKI Critically ill Patients

- Comorbidity-induced malnutrition
- Inflammation catabolism
- Muscle atrophy
- Immobility
- Anorexia
- Gut dysfunction
- Insulin resistance
- Anabolic resistance
- Uremic retention of toxic substances
- Inflammatory mediators
- Fluid retention
- Gut oedema
- Nutrient losses during RRT
- Caloric gain from citrate
- Catabolism
- Acute kidney injury
- Comorbidity-induced malnutrition
- Critical illness

Factors associated with PEW in Critically ill Patients

- Disease-related malnutrition (DRM) with inflammation
- Disease-related malnutrition (DRM) without inflammation
- Malnutrition/Undernutrition
- At risk for malnutrition

Every critically ill patient staying for more than 48 h in the ICU should be considered at risk for malnutrition. Expert
Medical nutrition therapy shall be considered for all patients staying in the ICU, mainly for more than 48 h. Good practice

References:
Factors associated with PEW in AKI Critically ill Patients

- Up to 42% of patients with AKI present with signs of severe malnutrition on admission*

Can we avoid PEW in AKI?

- Worsening of nutritional status is the result of many mechanisms:
  - inflammatory processes
  - catabolic factors
- Not a direct effect of nutrient intake

Nutrition can only improve protein and energy balance and possibly protein synthesis but cannot suppress critical illness-induced catabolism.
Nutritional and Dietary Management of Kidney Disease: A Patient-Centered Approach

Approach to Diet in AKD

- Energy (calories)
- Renal Minerals (potassium & phosphate)
- Protein
- Fluid & salt (sodium)
- Micronutrients: vitamins & minerals

Diet in Acute Kidney Disease

How much calorie to give?

- Energy expenditure (EE) tends to be higher during critical illness
- but both under- and overfeeding are associated with delayed recovery and increased mortality

**How to measure Energy Expenditure?**

**Resting energy expenditure**

- Indirect calorimetry
  - (O₂ consumption - CO₂ production)
  - calculate the total amount of energy produced

  **“Calculation”**
  - e.g. formula - Harris and Benedict:
    - Men (kcal): `REE = 66 + (13.7 × BW) + (5 × height) - (6.8 × age)`
    - Women (kcal): `REE = 65.5 + (9.6 × BW) + (1.8 × height) - (4.7 × age)`

- **“Estimation”**
  - (based on body weight, i.e. 25 kcal/kg/day)

**Indirect calorimetry: recommendations**

*In critically ill mechanically ventilated patients, EE should be determined by using indirect calorimetry.*  
**Grade B**


*A&A. We suggest that indirect calorimetry (IC) be used to determine energy requirements, when available and in the absence of variables that affect the accuracy of measurement.*

Energy Needs

Gold standard = indirect calorimetry

Clinical situation requiring careful interpretation of EE measured by indirect calorimetry:
- Agitation or unstable sedation
- Air leaks (10% min volume)
- Unstable body temperature (>1°C / 1 h)
- Unstable pH (0.1 +/- /1 h)
- FiO2 > 60%
- Organ support therapies – RRT, ECMO

How much calorie to give?

- Indirect calorimetry often not available
- Energy expenditure (EE) is difficult to predict

Based on expert consensus, in the absence of IC, we suggest that a predictive equation or a simplistic weight-based equation be used to determine Energy Requirements

→ 25-30 kcal/kg/day

- Equations failed in 80% of patients
  - equations overestimate calorific requirements
    - risks with over feeding well nourished
    - risks with under feeding poorly nourished
Energy Expenditure/Needs

Metabolism over time

Recommendation:
- Hypocaloric nutrition in the early phase of illness -> progressing slowly to full
- Targeted nutrition after day 3 with the aim to achieve more than 70% of EE -> but not more than 100%

How much calorie to give?

- There is no evidence that caloric targets should be different in AKI patients with and without RRT.
- In patients on CRRT, citrate contributes to caloric delivery and should be accounted for.

Nutritional and Dietary Management of Kidney Disease: A patient-Centered Approach

Caloric Recommendation

<table>
<thead>
<tr>
<th>Patients with AKI</th>
<th>Level of evidence *</th>
<th>General ICU patients</th>
<th>Level of evidence *</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gradual increase in first week</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o 20-30 kcal/kg/day</td>
<td>5</td>
<td>Gradual increase in first week to</td>
<td></td>
</tr>
<tr>
<td>o 25-30 kcal/kg/day</td>
<td>5</td>
<td>o 20-25 kcal/kg/day</td>
<td>2C</td>
</tr>
<tr>
<td></td>
<td></td>
<td>o up to 70% of EE in first 7 days</td>
<td>2C</td>
</tr>
</tbody>
</table>

1a: Systematic reviews (with homogeneity) of randomized controlled trials
2a: Systematic reviews (with homogeneity) of cohort studies
2b: Individual cohort study or low quality randomized controlled trials (e.g. <80% follow-up)
2c: “Outcomes” Research; ecological studies
5: Expert opinion without explicit critical appraisal, or based on physiological bench research or first principles

Osterman et. al., Intensive Care Medicine July 2019, Volume 45, Issue 7, pp 1006–1008

Calorie Intake and Patient Outcomes in Severe AKI: findings from the RENAL study trial

Analyzed Daily Calorie Intake in 1456 patients from the RENAL trial.

- Stable calorie intake was only achieved at 4 to 5 days after randomization
- Mean DCI was low at ~11 Kcal/Kg/day
  (not accounting for the CRRT caloric load from citrate and glucose)
- No difference in Mortality based on levels.
- In the multivariable analysis, high DCI levels was not independently associated with a significant decrease in the OR for 90-day mortality.

Bellomo at. al., Critical Care 2014, 18:R45
**How much protein to give?**

- More protein: beneficial or harmful?

1171 patients

*Zusman O et al Crit Care 2016;20:367*

**Mortality at D60**

**Effects of the nutrition regimen on urea appearance and protein catabolic rate**

- Few studies correlating of amount of nutrition received and the protein catabolic rate (PCR) measured in AKI patients
- Response to protein intake and restriction in AKI is consistent with the findings reported in other critically ill populations
- Decreased protein resulted in more muscle protein breakdown with the same generation of urea as those patients receiving moderate intakes of protein.
- Further increases in protein intake above 1.5 g protein/kg may lead to increased urea generation
- Increasing calories beyond energy expenditure may lead to increased protein breakdown and a more negative nitrogen balance.

*Mitchell H. Rosner, Nutritional Support for Patients with Acute Kidney Injury
NUTRITION ISSUES IN GASTROENTEROLOGY, SERIES #96, 2011*
**Recommendations: Protein Target**

**Guidelines for the Provision and Assessment of Nutrition Support Therapy in the Adult Critically Ill Patient: Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.)**

- Suggest that ICU patients with AKI be placed on a standard enteral formulation
  - protein (1.2–2 g/kg actual body weight per day)
  - energy (25–30 kcal/kg/d)
  - Consider a specialty formulation designed for renal failure (with appropriate electrolyte profile) if significant electrolyte abnormalities

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• Recommend patients receiving frequent hemodialysis or CRRT receive increased protein
  • Quality of Evidence: Very Low
  • Rationale:
    o A significant amino acid loss (10–15 g/d) in CRRT
    o Based on protein catabolic rate values estimated lean body mass catabolism ~ 1.4–1.8 g/kg/d in patients with AKI on CRRT
    o Additional 0.2 g/kg/d – max 2.5 g/kg/d
    o No advantages have been demonstrated with very high protein intakes (>2.5 g/kg/d)
    o Excessively high nitrogen intakes may simply increase the rate of urea production

• Protein should not be restricted in patients with renal insufficiency as a means to avoid or delay initiating dialysis therapy
### Protein Recommendation

<table>
<thead>
<tr>
<th>Patients with AKI</th>
<th>Level of evidence</th>
<th>General ICU patients</th>
<th>Level of evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AKI during critical illness and not on RRT:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o gradual increase to 1.3 g/kg/day</td>
<td>5</td>
<td>o progressive increase to 1.3 g/kg/day</td>
<td>2B</td>
</tr>
<tr>
<td>o up to 1.7 g/kg/day</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Critically ill patients on intermittent RRT:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o 1.0 - 1.5 g/kg/day</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o 1.5 g/kg/day</td>
<td></td>
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<td></td>
</tr>
<tr>
<td><strong>Critically ill patients on CRRT:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o up to 1.7 g/kg/day</td>
<td>5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2B: Individual cohort study or low quality randomized controlled trials (e.g., <80% follow-up)

2c: "Outcomes" Research; ecological studies

5: Expert opinion without explicit critical appraisal, or based on physiologic or bench research or first principles

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### Daily Protein Intake and Patient Outcomes in Severe AKI: Findings of the RENAL Trial

- **Mean DPI was low – 0.5g/kg/day**
  - Only 159 (10.9%) patients received a mean DPI of >1 g/kg
  - **26.8% of study days**
  - 200 patients received a combination of enteral and parenteral nutrition
  - **17.1% of study days**

- **Mortality**
  - DPI Below mean - 46%
  - DPI Above mean - 43%

- **Patients with a DPI above the median had similar mortality to patients with a below the median**
- **In the multivariable analysis, high DPI was not independently associated with a significant decrease in the OR for 90-day mortality**

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Bellomo at al., Blood Purif 2014;37:325–334
Nutritional and Dietary Management of Kidney Disease: A Patient-Centered Approach

Vitamins and trace elements

Recommendation

<table>
<thead>
<tr>
<th>Patients with AKI</th>
<th>General ICU patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>o Supplement micronutrient losses during extracorporeal treatment</td>
<td>o Routine supplementation with glutamine or 1b antioxidants not recommended</td>
</tr>
</tbody>
</table>
\[5\] | \[18\] |
| o Recommendation to detect micronutrient deficiencies in patient categories at risk  |                                    |
\[5\] |

1b: Individual randomized controlled trials (with narrow confidence interval)

5: Expert opinion without explicit critical appraisal, or based on physiology, bench research or "first principles"

Osterman et. al., Intensive Care Medicine July 2019, Volume 45, Issue 7, pp 1006–1008

Micronutrient Alterations During Continuous Renal Replacement Therapy in Critically Ill Adults: A Retrospective Study

Retrospective study Emory University Hospital’s

Between 2009 and 2012

75 patients receiving nutrition support services and had at least 1 serum micronutrient level measured during CRRT

Below normal range in:

- Thiamin 16% - (9/56)
- Pyridoxine 67% - (38/57)
- Ascorbic acid 37% - (13 of 15)
- Folate 33% - (3 of 9)
- Zinc 0% - (9 of 24)
- Copper 60% - (41 of 68)

- The incidence of various micronutrient deficiencies in critically ill patients who required CRRT was higher than previously reported.
- Prospective studies are needed to determine:
  - Is CRRT the main cause of low micronutrient status -?
  - the potential clinical and metabolic efficacy of supplementation in the intensive care unit setting.
Nutritional Screening post AKI

- In the study with 309 ARF patients admitted to the renal ward
- 67% of patients were dialyzed (206 of 309)
- **40% severe malnutrition** by Subjective Global Assessment (SGA)

Nutritional status of ARF patients on admission

In-hospital mortality according to nutritional status

- **All AKI patients with severe AKI should be screened for malnutrition** at discharge and at their first clinic appointment.

Fiaccadori et al., J AM Soc Nephrology, 1999

Fiaccadori and Cremaschi, Current Opinion in Critical Care 2009
Nutritional and Dietary Management of Kidney Disease: A Patient-Centered Approach

**Ideal Model for Nutrition Assessment**

- PO intake pre ICU hospital stay
- Micronutrients levels
- Recent weight loss
- BMI
- Body composition
- Acute inflammatory markers
  - IL-6
  - CRP
  - PCT
- Comorbid illness

**Tools**

- Malnutrition Universal Screening Tool (MUST)
- Nutritional Risk Screening (NRS 2002)
- Mini Nutritional Assessment (MNA)
- Short Nutritional Assessment Questionnaire (SNAQ)
- Malnutrition Screening Tool (MST)
- Subjective Global Assessment (SGA)
Malnutrition Universal Screening Tool (MUST)

Table 1 Initial screening

<table>
<thead>
<tr>
<th>Score</th>
<th>Question</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Is BMI ≥ 20.0?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Has the patient lost weight within the last 3 months?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Has the patient had reduced dietary intake in the last week?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Is the patient severely ill (e.g., in intensive therapy)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 2 Final screening

<table>
<thead>
<tr>
<th>Score</th>
<th>Impaired nutritional status</th>
<th>Severity of disease (± increase in requirements)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal nutritional status</td>
<td>Absent: Score 0 Normal nutritional requirements</td>
</tr>
<tr>
<td>Mid</td>
<td>Wt loss &gt;5% in 3 mths or Food intake below 70-75% of normal requirement in preceding week</td>
<td>Score 1 Hip fracture* Chronic patients, in particular with acute complications: Cancer*, COPD*, Chronic Arteriosclerosis, diabetes, oncology</td>
</tr>
<tr>
<td>2</td>
<td>Wt loss &gt;5% in 2 mths or BMI 18.5-20.5 = impaired general condition or Food intake 25-40% of normal requirement in preceding week</td>
<td>Moderate Score 2 Major abdominal surgery* Stroke* Severe pneumonia, hematologic malignancy</td>
</tr>
<tr>
<td></td>
<td>Wt loss &gt;5% in 1 mth (&gt;15% in 3 mths) or BMI &lt;18.5 = impaired general condition or Food intake 6-25% of normal requirement in preceding week</td>
<td>Severe Score 3 Head injury* Bone marrow transplantation* Intensive care patients (APACHE &gt; 15).</td>
</tr>
</tbody>
</table>

NUTrition Risk in the Critically Ill Score (NUTRIC Score)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Range</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>&lt;50</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>50 to &lt;75</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>≥75</td>
<td>2</td>
</tr>
<tr>
<td>APACHE II</td>
<td>&lt;15</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>15 to &lt;20</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>≥20</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>≥28</td>
<td>1</td>
</tr>
<tr>
<td>SOFA</td>
<td>&gt;6</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>6 to &lt;10</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>≥10</td>
<td>2</td>
</tr>
<tr>
<td>Number of comorbidities</td>
<td>0 to 1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>≥2</td>
<td>1</td>
</tr>
<tr>
<td>Days from hospital to ICU admission</td>
<td>9 to &lt;1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>≥1</td>
<td>1</td>
</tr>
<tr>
<td>Sum of points</td>
<td>Category</td>
<td>Points</td>
</tr>
<tr>
<td>5-9</td>
<td>High score</td>
<td>5</td>
</tr>
<tr>
<td>0-4</td>
<td>Low score</td>
<td>0</td>
</tr>
</tbody>
</table>

A high score is associated with higher 28-day mortality and longer duration of mechanical ventilation.

Subjective Global Assessment

- Incorporate information on functional status and physical examination
- Well nourished
- Moderately malnourished
- Severely malnourished

Most commonly used:

- History
  1. Weight (wt) change: In the past 2 weeks, weight has increased/decreased/not changed. Overall weight loss in the past 6 months: ________ kg. %.
  2. Change in dietary intake (relative to normal intake): circle No change, Borderline/Poor, Unable to eat. If intake has decreased, for how long? ________ weeks.
  3. Gastrointestinal symptoms (> 2 weeks): circle all that apply: None, Nausea, Vomiting, Diarrhea, Anorexia
  4. Functional capacity: circle No change, Decreased activities of daily living, Bed ridden
     - Metabolic stress: circle No stress, Low/moderate stress, High stress
     - Physical examination: check all that apply: Troops and chest subcutaneous fat loss, Quadriceps and deltoid muscle wasting

Nutritional and Dietary Management of Kidney Disease: A patient-Centered Approach

Heyland Critical Care 2011, 15:R28

UC San Diego Illescaspa
Nutritional Evaluation

1) CT Imaging Analysis

2) Quadriceps muscle layer thickness

3) Bioimpedance

Measuring Body Composition

Bioimpedance

Normally hydrated weight

- Total body water
- Intracellular water (ICW)
- Extracellular water (ECW)
- Body weight
- Fat Free Mass (FFM)
- Fat Mass (FM)
- Overhydration (OH)
- Lean Tissue Mass (LTM)
- Adipose Tissue Mass (ATM)

Nutritional and Dietary Management of Kidney Disease: A patient-Centered Approach
### Nutritional Assessment in AKI: Limitations

<table>
<thead>
<tr>
<th>Problems and limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Available bedside tools</td>
</tr>
<tr>
<td>Albumin, prealbumin, cholesterol</td>
</tr>
<tr>
<td>Lymphocyte count</td>
</tr>
<tr>
<td>TVD changes</td>
</tr>
<tr>
<td>Muscle wasting by anthropometry</td>
</tr>
<tr>
<td>PCR or protein equivalent of nitrogen appearance (PNA)</td>
</tr>
<tr>
<td>Energy expenditure (EE)</td>
</tr>
<tr>
<td>Nutritional scoring systems (SGA and its modifications)</td>
</tr>
<tr>
<td>Potential tools in development</td>
</tr>
<tr>
<td>Laboratory markers</td>
</tr>
<tr>
<td>Growth-hormone and IGF-1 levels</td>
</tr>
<tr>
<td>Inflammatory markers (PCR, serum interleukine levels, etc.)</td>
</tr>
<tr>
<td>Body mass and composition</td>
</tr>
<tr>
<td>Total body nitrogen</td>
</tr>
<tr>
<td>Energy-beam-based methods</td>
</tr>
<tr>
<td>Muscle fiber size and composition</td>
</tr>
<tr>
<td>Bioimpedance analysis</td>
</tr>
<tr>
<td>CT and/or MRI</td>
</tr>
</tbody>
</table>

May be low as negative markers of inflammation
Lack of specificity
Stable or increased TVD due to fluid gain even after body mass wasting
Less reliable if edema is present
In patients on RRT must be calculated by use kinetic methods
or directly measured after diuresis fluid reduction
Formulas for EE prediction not always reliable, especially in ill patients
Most available data in chronic renal failure patients

### Factors that May Affect Renal Recovery

- **Healthy kidney**
- **AKI**
- **DNA damage**
  - Increased age
  - Previous AKI/CKD
  - Sustained cell stress
- **CHD**
  - Progressive fibrosis

#### Risk factors
- **Age**
- Race or ethnic group
- Genetic factors
- Hypertension
- Diabetes mellitus
- Metabolic syndrome

#### Nutritional Status
- **Type and amount of calorie and protein intake**

#### Disease modifiers
- Severity of acute kidney injury
- Stage of chronic kidney disease
- Number of episodes
- Duration of acute kidney injury
- Proteinuria

#### Outcomes
- Cardiovascular events
- Kidney events
- End-stage renal disease
- Disability
- Diminished quality of life
- Death

Acute Kidney Injury: a springboard for progression in chronic kidney disease

**INCOMPLETE RECOVERY POST-AKI**
- Fibrosis and Maladaptive repair
- Vascular drop out as a consequence of endothelial injury
- Nephron loss followed by glomerular hypertrophy

**REDUCED KIDNEY MASS**
- Hyperfiltration in remaining nephrons
- Increase epithelial transport
- Higher oxidative stress

**TUBULAR INTERSTITIAL FIBROSIS**


**Experimental Studies**

**DIETARY PROTEIN RESTRICTION**

**REDUCE INTRA-GLomerular PRESSURE**

**CHANGE CYTOKINE EXPRESSION**

Reduce glomerular scarring
And matrix deposition

**SLOW RATE OF RENAL FUNCTION DECLINE**

Venkatachalam et al, Am J Physiol Renal Physiol 298, 2010
Metabolic acidosis may be also implicated in the association of diet and kidney disease progression.

Metabolic Acidosis and Progression to Chronic Kidney Disease

Dietary acid load, even in the absence of overt acidosis, may have deleterious effects.

Increased ammonia generation per nephron

Metabolic acidosis/High dietary acid intake

↑ Single nephron ammoniagenesis

↑ Endothelin-1

↑ Aldosterone

Alternative complement pathway

Tubulo-interstitial injury

Glomerulosclerosis

Decline in renal function

Chen and Abramowitz BMC Nephrology 2014, 15:55
Dietary acid load and chronic kidney disease among adults in the United States

Study cohort (n=12293): cross-sectional analysis of adult participants of the National Health and Nutrition Examination Survey (NHANES) 4.

After multivariable adjustment for demographics, CKD risk factors, caloric intake, and BMI, greater Net Acid Excretion was associated with reduced eGFR. The highest quintile of NAE was associated with 1.4 fold greater odds of low eGFR compared with the lowest.

Do NOT COPY
Summary

- Malnutrition is a frequent complication during and post AKI.
- Several factors contribute to the development of malnutrition, mainly the degree of inflammatory status and severity of comorbidities.
- Tools for the assessment of nutritional status need to be validated.
- The amount, type and timing of nutritional support is not defined during or post the AKI episode.
- The effect of nutrition in the recovery of renal function still needs to be established.

Thank you